Articles

Effect of passive smoking on respiratory symptoms, bronchial responsiveness, lung function, and total serum IgE in the European Community Respiratory Health Survey: a cross-sectional study

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Summary

Background Passive smoking is widespread, and environmental tobacco smoke contains many potent respiratory irritants. This analysis aimed to estimate the effect of passive smoking on respiratory symptoms, bronchial responsiveness, lung function, and total serum IgE in the European Community Respiratory Health Survey.

Methods This analysis included data from 7882 adults (age 20–48 years) who had never smoked, from 36 centres in 16 countries. Information on passive smoking, respiratory symptoms, asthma, and allergic rhinitis was gathered through a structured interview. Spirometry and methacholine challenge were carried out, and total and specific IgE were measured. The effect of passive smoking was estimated by means of logistic and multiple linear regression for each country and combined across countries by random-effects meta-analysis.

Findings In 12 of the 36 centres, more than half the participants were regularly involuntarily exposed to tobacco smoke. The prevalence of passive smoking in the workplace varied from 2·5% in Uppsala, Sweden, to 53·8% in Galdakao, Spain. Passive smoking was significantly associated with nocturnal chest tightness (odds ratio 1·28 [95% CI 1·02 to 1·60]), nocturnal breathlessness (1·30 [1·01 to 1·67]), breathlessness after activity (1·25 [1·07 to 1·47]), and increased bronchial responsiveness (effect −0·18 [−0·30 to −0·05]). Passive smoking in the workplace was significantly associated with all types of respiratory symptoms and current asthma (odds ratio 1·90 [95% CI 0·90 to 2·88]). No significant association was found between passive smoking and total serum IgE.

Interpretation Passive smoking is common but the prevalence varies widely between different countries. Passive smoking increased the likelihood of experiencing respiratory symptoms and was associated with increased bronchial responsiveness. Decreasing involuntary exposure to tobacco smoke in the community, especially in workplaces, is likely to improve respiratory health.

Lancet 2001; **358:** 2103–09

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Introduction

Passive smoking is widespread and causes exposure to many potent respiratory irritants.[1,2] In children, parental smoking is associated with respiratory symptoms and poorer lung function.[3] The consequences of involuntary exposure to tobacco smoke have not been studied as extensively in adults as in children.[4] Some studies have found that passive smoking in adulthood increases the risk and severity of asthma and respiratory symptoms.[5–7] Several studies have indicated that involuntary exposure to tobacco in adults results in significant impairment of lung function.[8–10] Passive smoking has also been associated with increased serum concentrations of total IgE.[11,12]

Between 1990 and 1994, information on variation in asthma prevalence, known or suspected risk factors for atopy and asthma, and the management of asthma were collected in the European Community Respiratory Health Survey (ECRHS).[13] The association between passive smoking and respiratory symptoms and asthma has so far been analysed in only one ECRHS study, which was based on data from the Swedish part of the survey; passive smoking in the workplace was found to increase the risk of symptomatic asthma in the workplace and asthma-related work disability.[14]

The aim of this analysis was to study the effect of passive smoking on respiratory symptoms, bronchial responsiveness, lung function, and total serum IgE in the ECRHS.

Population and methods

Population

The methods of the ECRHS have been described elsewhere.[13] Participating centres selected an area defined by existing administrative boundaries with a population of at least 150 000. When possible, an appropriate local sampling frame was used to select randomly at least 1500 men and 1500 women, aged 20–44 years. In stage I, the ECRHS screening questionnaire was sent to participants. It asked about symptoms suggesting asthma, the use of medication for asthma, and the presence of hay-fever and nasal allergies. In stage II, which took place in 1990–94, a smaller random sample of participants who had completed the screening questionnaire were invited to attend for a more detailed interview-led questionnaire, blood tests for the measurement of total and specific IgE, spirometry, and methacholine challenge. This analysis

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included all 36 centres with both questionnaire and IgE data.

Design and procedures

The interviewer-led questionnaire included, among other items, questions on respiratory symptoms, self-reported asthma and allergic disorders, and environmental and lifestyle factors. The participants were asked whether they had experienced the following symptoms in the previous 12 months: wheezing or whistling in the chest; wheezing in combination with breathlessness; wheezing when not affected by a cold; being woken by a feeling of tightness in the chest; an attack of shortness of breath during the day when resting; an attack of shortness of breath after strenuous activity; or being woken by an attack of shortness of breath.

Current asthma was defined as answering "yes" to the question "Have you ever had asthma?" and having had an attack of asthma in the previous 12 months. Allergic rhinitis was defined as answering "yes" to the question: "Do you have hay-fever or any other kind of allergic rhinitis?" The participants were asked whether they had smoked at least one cigarette per day or one cigar per week for 1 year or 360 g tobacco in their lifetime. Current smoking was defined as answering "yes" to the question: "Do you now smoke?" Individuals who had stopped smoking less than 12 months before the examination were defined as current smokers.[15] Except when we were estimating the prevalence of current smoking within each centre, all analyses included only those participants who reported that they had never smoked.

Parental smoking was defined as an affirmative answer to either of the following questions: "Did your father ever smoke regularly during your childhood?" and "Did your mother ever smoke regularly during your childhood or before you were born (during pregnancy)?". Passive smoking was assessed by use of different items from the questionnaire. The participants were asked whether they had regularly (most days or nights) been exposed to tobacco smoke in the previous 12 months. Participants answering "yes" to this question were classified as being passive smokers and then asked the number of other people in the household who regularly smoked. Participants with at least one smoker in the household were classified as involuntarily exposed to tobacco smoke in the household. Participants were also asked whether people smoked regularly in the room where they worked, and those who answered "yes" were classified as being passive smokers in the workplace. Participants were then asked to estimate how many hours per day they were exposed to other people's tobacco smoke.

A job exposure matrix was used to assess occupational exposures in the current or last-held job.[16] The matrix was developed for the ECRHS, and consisted of 350 occupational groups on the job axis, and a semiquantitative estimate of exposure (none, low, or high) separately for biological dust, mineral dust, and gases or fumes on the exposure axis.

Forced expiratory volume in 1 s (FEV_1) and forced vital capacity (FVC) were recorded by a standard spirometric method.[17] The ratio between FEV_1 and FVC was calculated. FEV_1, as a percentage of the predicted value, was calculated by the formula from the European Community for Steel and Coal.[18] Methacholine challenge was done with a dosimeter (Mefar, Brescia, Italy). The degree of bronchial responsiveness was expressed on the

Centre and country	Total participants	Proportion exposed to passive smoking (%)	Proportion exposed to passive smoking at home (%)	Number employed	Proportion exposed to passive smoking at work (%)	Proportion exposed to parental smoking (%)
Galdakao (Spain)	170	75·9	50·6	130	53·8	60·6
Huelva (Spain)	101	67·3	41·6	72	48·6	80·2
Pavia (Italy)	123	61·8	35·8	103	41·8	62·1
Oviedo (Spain)	107	56·1	28·0	73	41·1	54·2
Bergen-op-Zoom (Netherlands)	166	59·0	28·3	146	37·7	78·9
Barcelona (Spain)	140	55·0	28·6	119	36·1	57·4
Geleen (Netherlands)	144	56·9	23·6	126	34·9	79·3
Albacete (Spain)	151	60·9	36·4	90	32·2	64·9
Turin (Italy)	130	55·4	30·8	122	31·2	70·8
Verona (Italy)	155	45·2	24·5	126	30·2	64·5
South Antwerp (Belgium)	261	49·8	21·1	239	30·1	70·0
Groningen (Netherlands)	141	38·3	17·0	100	29·4	75·5
Hamburg (Germany)	402	45·3	18·2	337	29·4	61·7
Dublin (Ireland)	171	63·2	42·1	161	29·2	76·9
Antwerp City (Belgium)	218	47·7	21·6	201	28·4	64·7
Bordeaux (France)	201	52·7	21·9	123	27·6	68·2
Erfurt (Germany)	260	35·8	10·8	246	24·8	53·6
Ipswich (UK)	234	36·3	13·7	186	24·2	65·8
Paris (France)	246	44·3	12·6	223	23·3	60·3
Basel (Switzerland)	352	29·6	13·6	282	20·2	52·4
Norwich (UK)	226	34·1	15·4	193	19·7	68·4
Bergen (Norway)	344	32·0	18·3	288	19·1	65·2
Montpellier (France)	218	37·2	13·8	195	18·0	59·2
Reykjavik (Iceland)	213	53·0	23·0	156	18·0	63·2
Grenoble (France)	197	33·5	15·7	186	17·7	64·5
Cardiff (UK)	195	39·0	19·6	190	13·7	76·2
Tartu (Estonia)	208	33·6	20·3	169	13·0	60·0
Cambridge (UK)	155	31·6	14·3	142	11·3	67·7
Göteborg (Sweden)	293	38·2	15·4	263	10·3	63·8
Hawkes Bay (New Zealand)	168	29·2	12·5	166	10·2	64·9
Umeå (Sweden	291	18·9	8·6	247	8·9	61·9
Melbourne (Australia)	329	20·7	11·6	316	7·9	64·1
Portland (USA)	411	20·7	10·0	309	7·1	64·9
Wellington (New Zealand)	238	24·4	13·9	202	5·9	68·4
Christchurch (New Zealand)	202	21·8	12·5	178	5·1	75·1
Uppsala (Sweden)	321	21·5	9·4	278	2·5	60·1

Centres are ranked according to frequency of passive smoking in the workplace.

Table 1: **Prevalence of passive smoking by centre**

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ECRHS slope.[19,20] Height was measured before measurement of lung function.

Total and specific serum IgE was measured with the Pharmacia CAP System (Pharmacia Diagnostics, Uppsala, Sweden). In all centres, we measured specific IgE against antigens of *Dermatophagoides pteronyssinus*, timothy grass, cat, and *Cladosporium herbarum*. Detection of specific IgE (>0·35 kU/L) was used as the definition of sensitisation. Atopy was defined as being sensitised to any of the allergens above.

Statistical analysis

The statistical analysis used Stata (version 6.0). Logistic regression was used to calculate odds ratios for the effect of passive smoking on respiratory symptoms, asthma, and allergic rhinitis. Multiple linear regression was used to analyse the effect of passive smoking on lung function, bronchial responsiveness, and total IgE. The adjusted odds ratios and regression coefficients were estimated separately for each country from individual data, with adjustment for centre within country. Average effect estimates were derived, and potential heterogeneity between countries was examined by a standard method for random-effects meta-analysis.[21] This method is equivalent to a multilevel model with level 1 as individual and level 2 as country, with the effect of each independent factor allowed to vary between countries. In some of the analyses, one, two, or three of the countries had to be excluded owing to insufficient data. The dose-related effect of passive smoking on respiratory symptoms, lung function, bronchial responsiveness, and total serum IgE was assessed by division of the duration of reported daily passive smoking into four categories (less than 1 h, 1 h to less than 4 h, 4 h to less than 8 h, and 8 h or longer). These analyses were done by logistic regression and multiple linear regression on the pooled dataset with adjustment for centre. Associations between passive smoking in the workplace and respiratory outcomes were also adjusted for concurrent occupational exposures, by entering the variables of any low and any high exposure according to the job exposure matrix in the model.

Results

The analysis included data from 36 centres in 16 countries. No data on bronchial responsiveness were available for two of the centres (Cardiff and Tartu). Altogether we included 3486 men and 4396 women (mean age 32·7 years [range 20–48]) who had never smoked. Of the 7882 questionnaire respondents included, spirometry was done for 6655, IgE measurements for 5985, and methacholine challenge for 5403. Data on occupational exposures in the current or last-held job were available from 30 of the 36 centres (5321 participants).

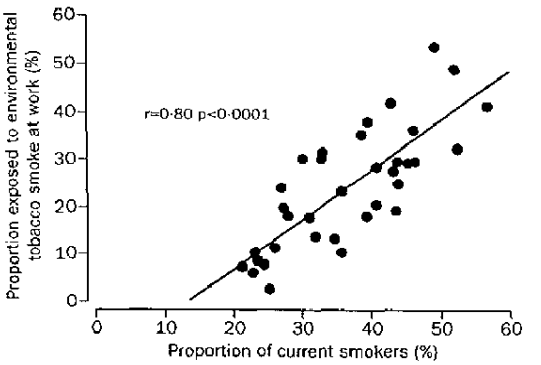

Figure 1: **Ecological analysis of association between prevalence of smoking in all participants in a centre and frequency of passive smoking in the workplace in occupationally active participants**

In 12 of the 36 centres, more than half the population reported that they were passive smokers (table 1). The highest prevalence of passive smoking in the workplace among occupationally active individuals was in Galdakao, Spain, and the lowest in Uppsala, Sweden. In an ecological analysis, a positive correlation was found between the prevalence of current smoking in the whole population in each centre and the prevalence of passive smoking in the workplace (figure 1). A significant ecological association was also found between the community prevalence of smoking and passive smoking (r=0·77) and passive smoking in the household (r=0·70, p<0·0001).

Multivariate analysis showed that passive smoking was significantly associated with nocturnal chest tightness and breathlessness, attacks of shortness of breath after strenuous activity, and being woken by an attack of shortness of breath (table 2). In the analyses, adjustments were made for age, sex, parental smoking, allergic sensitisation, and total serum IgE. No association was found between passive smoking and current asthma or allergic rhinitis. There was significant heterogeneity between countries in the relation between passive smoking and wheeze; a positive association was found in most countries, but there was a trend towards a negative association in Estonia, Ireland, the UK, and New Zealand (figure 2).

	Passive smoking, all		Passive smoking at home		Passive smoking at work	
	Odds ratio (95% CI)*	p for heterogeneity	Odds ratio (95% CI)†	p for heterogeneity	Odds ratio (95% CI)‡	p for heterogeneity
Wheeze	1·28 (0·99–1·65)	0·01	0·84 (0·67–1·05)	0·16	1·50 (1·20–1·88)	0·62
Wheeze and breathlessness	1·14 (0·88–1·49)§	0·14	0·86 (0·65–1·14)§	0·68	1·45 (1·10–1·92)§	0·74
Wheeze without cold	1·19 (0·90–1·58)§	0·11	0·86 (0·64–1·15)§	0·67	1·44 (1·02–2·02)§	0·21
Nocturnal chest tightness	1·28 (1·02–1·60)	0·17	1·00 (0·79–1·26)	0·74	1·60 (1·14–2·25)	0·04
Breathless when resting	1·08 (0·74–1·58)	0·02	1·00 (0·66–1·52)§	0·15	1·45 (1·00–2·25)§	0·28
Breathless after effort	1·25 (1·07–1·47)	0·47	1·08 (0·88–1·32)	0·64	1·31 (1·06–1·62)	0·47
Nocturnal breathlessness	1·30 (1·01–1·67)	0·61	1·17 (0·84–1·62)§	0·59	1·56 (1·12–2·18)§	0·44
Current asthma	1·15 (0·84–1·58)	0·43	1·14 (0·68–1·90)§	0·17	1·90 (1·25–2·88)§	0·98
Allergic rhinitis	1·02 (0·81–1·20)	0·30	0·95 (0·77–1·16)	0·33	1·18 (0·90–1·55)	0·06

*Adjusted for age, sex, parental smoking, sensitisation to mite, cat, timothy grass, and cladosporium antigens, total IgE, and centre. †Adjusted for same variables as passive smoking, all, and for passive smoking at work. ‡Adjusted for same variables as passive smoking, all, and for passive smoking at home. §One, two or three countries left out of the analysis because of insufficient data.

Table 2: **Odds ratios for respiratory symptoms, current asthma, and allergic rhinitis in passive smokers compared with participants without regular involuntary exposure to tobacco smoke**

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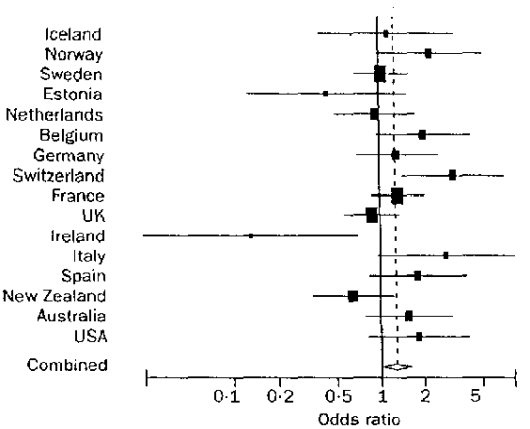

Figure 2: **Adjusted odds ratios for wheeze in never-smoking individuals with exposure to tobacco smoke compared with individuals without regular exposure**

Adjusted within countries for age, parental smoking, allergic sensitisation, total serum IgE, and centre. For each country, horizontal line indicates 95% CI. For combined odds ratio, diamond indicates 95% CI, from the model with country as the random effect. The size of each square is proportional to the sample size.

There were no significant associations between passive smoking in the household and respiratory symptoms (table 2). Passive smoking in the workplace was significantly related to current asthma and to all respiratory symptoms except attacks of breathlessness at rest, but no relation was found between passive smoking in the workplace and allergic rhinitis (table 2). With adjustment for other occupational exposures, no significant association was found between passive smoking in the workplace and attacks of nocturnal breathlessness (odds ratio 1·46 [95% CI 0·97–2·21]). All other associations remained significant. The odds ratios for wheeze and current asthma were 1·59 (1·23–2·07) and 2·73 (1·58–4·74) with adjustment for other occupational exposures.

A significant dose-related association with passive smoking was found for all respiratory symptoms (table 3). Passive exposure to tobacco smoke for at least 8 h per day significantly increased the risk of all respiratory symptoms except attacks of nocturnal breathlessness compared with individuals without regular exposure. No significant dose-related association was found between passive smoking and current asthma or allergic rhinitis.

Passive smoking was related to increased bronchial responsiveness (decreased ECRHS slope) (table 4). The association between bronchial responsiveness and both passive smoking in general and passive smoking in the home remained significant when adjustment was made for FEV_1 expressed as a percentage of the predicted value (estimated changes in slope value –0·14 [95% CI–0·27 to –0·01] and –0·18 [–0·33 to –0·03]). No significant association was found between any type of passive smoking and total serum IgE or lung function (table 4).

Significant dose-related trends were found between passive smoking and bronchial responsiveness and FEV_1 (table 5). The dose-related association was due mainly to the difference between exposure of less than 1 h and longer durations. No significant dose-related relation was found between passive smoking and total serum IgE.

The possibility of a difference between the sexes in the effect of passive smoking was investigated by testing the interaction term. No sex difference was found in the association between passive smoking and any symptom, bronchial responsiveness, total serum IgE, or lung function. Nor was there evidence that the associations with passive smoking differed between atopic and non-atopic individuals or between those who had been exposed or not exposed to parental smoking (p for all interactions tested >0·1).

Discussion

The main findings in this investigation of adults who had never smoked are that reported passive smoking was common in all centres. However, the prevalence of passive smoking in the workplace varied more than 20-fold, with high rates in southern and central Europe and lower rates in New Zealand, Australia, the USA, and

	1 to <4 h (n=1246)	4 to <8 hours (n=565)	≥8 h (n=447))	p for trend
Wheeze	1·11 (0·91–1·35)	1·39 (1·08–1·80)	1·49 (1·12–1·97)	0·001
Wheeze and breathlessness	1·07 (0·84–1·37)	1·27 (0·93–1·73)	1·47 (1·04–2·07)	0·02
Wheeze without cold	0·95 (0·74–1·23)	1·51 (1·10–2·08)	1·75 (1·24–2·45)	<0·001
Nocturnal chest tightness	1·12 (0·91–1·39)	1·43 (1·09–1·89)	1·59 (1·18–2·13)	<0·001
Breathless when resting	0·64 (0·46–0·89)	1·38 (0·97–1·96)	1·82 (1·26–2·63)	0·005
Breathless after effort	1·15 (0·95–1·38)	1·30 (1·02–1·66)	1·46 (1·12–1·91)	0·001
Nocturnal breathlessness	1·11 (0·83–1·49)	1·28 (0·88–1·86)	1·42 (0·95–2·12)	0·048
Current asthma	0·99 (0·70–1·40)	1·19 (0·76–1·88)	1·39 (0·86–2·25)	0·18
Rhinitis	1·00 (0·84–1·18)	0·99 (0·78–1·25)	0·93 (0·71–1·20)	0·64

*Adjusted for age, sex, parental smoking, sensitisation to mite, cat, timothy grass, and cladosporium antigens, total IgE, and centre.

Table 3: **Odds ratios (95% CI) for respiratory symptoms, current asthma, and allergic rhinitis in passive smokers categorised by daily exposure duration compared with participants without regular involuntary exposure to tobacco smoke (<1 h daily)**

	Passive smoking, all		Passive smoking at home*		Passive smoking at work†	
	Effect (95% CI)	p for heterogeneity‡	Effect§ (95% CI)	p for heterogeneity‡	Effect (95% CI)	p for heterogeneity
IgE (log_{10} kU/L)§	–0·02 (–0·05 to 0·02)	0·31	–0·01 (–0·06 to 0·03)	0·39	–0·02 (–0·06 to 0·03)	0·52
ECRHS slope¶	–0·18 (–0·30 to –0·05)	0·38	–0·18 (–0·33 to –0·03)	0·99	–0·06 (–0·23 to 0·10)	0·35
FEV_1 (mL)‡	–23 (–49 to 2·7)	0·63	–8·3 (–42 to 25)	0·78	–5·1 (–50 to 40)	0·08
FVC (mL)‡	–22 (–51 to 8·2)	0·48	–5·9 (–44 to 32)	0·50	–0·7 (–40 to 39)	0·59
FEV_1/FVC (%)‡	–0·11 (–0·46 to 0·24)	0·41	0·01 (–0·42 to 0·43)	0·67	–0·04 (–0·56 to 0·49)	0·20

*Effect adjusted for passive smoking at work as well as variables listed for each characteristic. †Effect adjusted for passive smoking at home as well as variables listed for each characteristic. ‡Also adjusted for total serum IgE, height, and interaction of sex with height and age. §Adjusted for age, sex, parental smoking, sensitisation to mite, cat, timothy grass, and cladosporium antigens, and centre. ¶Also adjusted for total serum IgE.

Table 4: **Estimated effect of passive smoking on total serum IgE, bronchial responsiveness (ECRHS slope), and lung function**

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Source: https://www.industrydocuments.ucsf.edu/docs/tnjj0001

	Effect (95% CI)			
	1 to <4 h (n=1220)	4 to <8 h (n=555)	≥8 h (n=401)	p for trend
IgE (log_{10}(kU/L))*	−0·03 (−0·07 to 0·01)	−0·00 (−0·06 to 0·05)	−0·02 (−0·08 to 0·04)	0·37
ECRHS slope (units)†	−0·21 (−0·35 to −0·06)	−0·15 (−0·35 to 0·05)	−0·20 (−0·42 to 0·01)	0·01
FEV1 (mL)‡	−35 (−66 to −4·1)	−9·3 (−52 to 33)	−63 (−111 to −15)	0·01
FVC (mL)‡	−29 (−65 to 6·4)	−2·1 (−70 to 29)	−41 (−96 to 14)	0·08
FEV_1/FVC (%)‡	−0·27 (−0·68 to 0·15)	0·14 (−0·44 to 0·71)	−0·61 (−1·25 to 0·04)	0·18

*Adjusted for age, sex, parental smoking, sensitisation to mite, cat, timothy grass, and cladosporium antigens, and centre. †Also adjusted for total serum IgE. ‡Also adjusted for total serum IgE, height, and interaction of sex with height and age.

Table 5: **Estimated effect (95% CI) on total serum IgE, bronchial responsiveness (ECRHS slope) and lung function in passive smokers categorised by daily exposure duration compared with participants without regular involuntary exposure to tobacco smoke (<1 h/daily)**

Sweden. Passive smoking increased the likelihood of experiencing respiratory symptoms and was also related to increased bronchial responsiveness. We found a positive association between passive smoking in the workplace and current asthma.

Our ecological analyses indicate that the geographical variation in passive smoking is mainly related to the community prevalence of smoking. Differences in legislation and compliance with legislation between countries may also explain some of the variation in passive smoking in the workplace. A low community prevalence of smoking probably also facilitates legislation against involuntary tobacco exposure in the workplace. Data from a recent WHO report show that during the 1990s the prevalence of tobacco smoking decreased in some countries, such as the UK, France, and Sweden, whereas it remained stable in countries like Spain and Italy.[22] This information suggests that the current geographical variation in the prevalence of passive smoking may be even greater than that observed in our study.

In our investigation, passive smoking was positively associated with respiratory symptoms. Other population studies have shown that individuals who are exposed to environmental tobacco smoke are more likely to report respiratory symptoms.[6,23] The odds ratio for the relation between passive smoking and wheeze was lower than in a study of a Swiss population sample.[6] In our study the adjusted odds ratio for the association between passive smoking and wheeze was 2·8 (1·3 to 6·3) in Basel, Switzerland, which is also higher than in the study overall, and there may be true heterogeneity between countries in the associations with wheeze as suggested in table 2. We found a dose-dependent relation between the amount of involuntary tobacco exposure expressed as hours per day and the likelihood of reporting respiratory symptoms, as did Leuenberger and colleagues.[6]

Our investigation also indicates that the location of passive smoking may be of importance. Passive smoking in the workplace was strongly associated with respiratory symptoms, but no significant association was found for exposure in the household. This finding accords with those of Leuenberger and colleagues.[6] The wording of the questions related to passive smoking may have partly caused the discrepancy between workplace and household exposure. Passive smoking in the household was in our investigation defined as having at least one person at home who smokes, whereas passive smoking in the workplace was defined as having people who smoke regularly in the room where you work. An individual who lives with someone who is a smoker but who does not smoke inside the house could, in our investigation, be defined as being involuntarily exposed to tobacco in the household. However, a more likely explanation of the lack of association between passive smoking at home and symptoms is that other family members are more likely to stop smoking if one of their family develops respiratory symptoms.

In one prospective study, passive smoking was found to increase the risk of developing asthma.[5] An association between passive smoking and self-reported asthma has also been found in cross-sectional studies.[6,7] In our investigation, current asthma was strongly associated with passive smoking in the workplace, in accordance with the previous analysis of data from the three Swedish centres in the ECRHS.[14] This association remained significant after adjustment for other occupational irritants such as dusts, gases, and fumes, which suggests that the asthma risk associated with workplace tobacco exposure was not caused by other obvious concomitant workplace exposures. No significant association was, however, found between overall involuntary tobacco exposure or household exposure and asthma. We could not find any relation between the doses of involuntary tobacco exposure and the likelihood of reporting current asthma. Like Leuenberger and colleagues, we found no association between passive smoking and allergic rhinitis.[6]

In this study, passive smoking was associated with increased bronchial responsiveness. To our knowledge, the association between passive smoking and bronchial responsiveness in adults has not previously been assessed in studies of the general population. Exposure to cigarette sidestream smoke in provocation tests has, however, been shown to lead to an acute decline in lung function and increased bronchial responsiveness in some individuals.[24,25]

Some, but not all, studies have shown that passive smoking is associated with a small but significant impairment in lung function.[6–10,26,27] In this analysis, passive smokers had lower lung function than those who were not exposed, although the differences did not achieve significance. There was, however, a significant dose-related trend between passive smoking and FEV_1, and participants who were exposed to other people's tobacco smoke for 8 h or more per day showed significantly poorer lung function than those who were not exposed at all.

Active smoking is associated with increased concentrations of total serum IgE,[11,12] and a previous report from the ECRHS has observed such an association in this study population.[28] In this investigation, we could not find any significant relation between passive smoking and total serum IgE. An association between passive smoking and increased concentrations of total serum IgE was reported from two population studies.[11,12] We cannot explain why our study differs in this respect from previously reported studies. Our results, however, accord with those of Strachan and Cook,[29] who in a systematic review reported that there was no significant association of maternal smoking with concentrations of total serum IgE in neonates or in older children, when this association was analysed by meta-analysis.

Some of the previously published studies have shown that the effect of passive smoking may differ between

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women and men.[7,9,12] In our study, however, no sex difference was found in the association between passive smoking and any symptom, bronchial responsiveness, total serum IgE, or lung function, when we used a test of multiplicative interaction. There are indications that the effect of active smoking differs between atopic and non-atopic people.[30,31] We did not find, however, that the effect of passive smoking differed significantly between allergen-sensitised and non-sensitised individuals. Nor were there significant differences in the effect of passive tobacco smoking in individuals who had been exposed or not exposed to parental smoking as children.

There are some important limitations in our study, which should be taken into account when interpreting the results. The most important is that except in one centre we did not include objective measurements of passive smoking, such as salivary, serum, or urine cotinine concentrations. However, in one centre (Verona, Italy), non-smoking participants in the ECRHS who were defined as passive smokers by the ECRHS questionnaire had serum cotinine concentrations about twice those of non-exposed participants. A significant correlation was found between duration of exposure to tobacco smoke and serum cotinine concentrations.[32] Previous studies have shown a modest correlation between self-reported passive smoking and biomarkers of tobacco exposure.[1,33,34] Reliance on self-reported data does, however, carry a risk of misclassification, because individuals with symptoms may be more likely to report involuntary tobacco exposure than those without symptoms. A second limitation is that the study is cross-sectional, and we have no data on the duration of tobacco exposure. The strengths of this investigation are that it is one of the largest surveys to date of the effect of passive smoking on respiratory health and it has investigated both subjective effect markers, such as symptoms and reported asthma, and objective measurements, such as lung function and bronchial responsiveness.

Decreasing involuntary exposure to tobacco smoke in the community, especially in workplaces, is likely to improve respiratory health.

Contributors

C Janson wrote the report and analysed data. S Chinn collated data and developed the statistical model for data analysis. D Jarvis participated in overall design of the study. J-P Zock collated data concerning the job exposure matrix. K Torén analysed the association between passive smoking and occupational asthma in a previous report on the Swedish subsample of the data set. P Burney was responsible for the overall design of the study. All authors helped to write the report.

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Acknowledgments

The coordination of this work was supported by the European Commission. We thank Colette Baya and Manuel Hallen for their help during the study and K Vuylsteek and the members of the COMAC for their support. The following grants helped to fund the local studies: Allen and Hanbury's, (Australia); Belgian Science Policy Office, National Fund for Scientific Research (Belgium); Estonian Scientific Foundation (grant 1088), Glaxo Wellcome (Estonia); Ministere de la Santé, Glaxo France, Institut Pneumologique d'Aquitaine, Contrat de Plan Etat-Région Languedoc-Rousillon, CNMATS, CNMRT (90MR/10, 91AF/6), Ministre delegué de la santé, RNSP (France); GSF, and the Bundesminister für Forschung und Technologie, Bonn (Germany); Ministero dell'Università e della Ricerca Scientifica e Tecnologica, CNR, Regione Veneto grant RSF number 381/05.93 (Italy); Asthma Foundation of New Zealand, Lotteries Grant Board, Health Research Council of New Zealand (New Zealand); Norwegian Research Council project number 101422/310 (Norway); Ministero Sanidad y Consumo FIS grants 91/0016060/00E-05E, 92/0319, 93/0393, Hospital General de Albacete, Hospital General Juan Ramón Jiménez, Consejeria de Sanidad Principado de Asturias (Spain); Swedish Heart Lung Foundation, Swedish Medical Research Council, Swedish Association against Asthma and Allergy (Sweden); Swiss National Science Foundation grant 4026-28099 (Switzerland); National Asthma Campaign, British Lung Foundation, Department of Health, South Thames Regional Health Authority (UK); United States Department of Health, Education and Welfare Public Health Service Grant 2 S07 RR05521-28 (USA).

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Uses of error: Examination

A 37-year-old engineer from Belarus was admitted to our hospital for aortic valve replacement. We had received the results of his echocardiography several months beforehand, which showed severe aortic incompetence due to a bicuspid valve, with left ventricular and aortic root dilation. I went to the ward to see the patient with a bilingual medical student translating for me. I examined the patient carefully, finding some characteristics of Marfan's disease without completely fulfilling the necessary criteria. My suspicion of Ehlers Danlos syndrome was later confirmed by the intraoperative skin biopsy. With some pride I demonstrated all the physical findings in aortic regurgitation, showing the student how to auscultate from the anterior chest wall. I omitted auscultation of the intrascapular region. The transoesophageal echocardiogram was repeated on admission, as part of a routine preoperative work-up. Our echographist showed the bicuspid aortic valve, but thought that the descending aorta looked suspicious. We then did magnetic resonance imaging of the chest and found that the patient also had coarctation of the aorta. The pressure gradient was 100 mm Hg. There are three lessons to be learnt from this experience; 1) clear physical signs, such as a loud intrascapular murmur, may be missed over decades of repeated examinations. 2) Even in times of cost-consciousness it is sometimes better to repeat a diagnostic procedure. 3) We must examine the patient as thoroughly and meticulously as we insist our students do.

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